vent its sporulation and thus cut short the ague paroxysms. Cases have been reported[17] by Mya, Thayer, Boinet, Thintignan, Huddleston, of New York, and Mühll, of Basle, and others. If methylene blue stains the parasite or its nucleus, this blue, commingling with the red of the blood, would produce the (to the parasite) disastrous violet or purple.

Possibly the (hitherto enigmatical) curative action of sulphate of quinine may be due to its remarkable *fluorescence*. In solution it intensifies the violet, and even renders the ultra-violet rays of the spectrum perceptible to human vision.

Of the few vegetable products having this quality of fluorescence, another one is esculin, the bitter principle of the horse-chestnut tree bark (*Æsculus hippocastanum*). But this also has been successfully used as an antiperiodic for intermittent fever.[18] Do these fluorescent substances act by intensifying violet rays in the blood?

If the etiology now given be correct, treatment is self-evident, viz., keep malarial patients in the dark, or in rooms with purple or indigo windows, and clothe them with garments impenetrable to light; in the tropics, with white clothing lined with purple or black. Give drugs that darken the blood or render it violet, or lessen its translucency.

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ANGINA PECTORIS.

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ANGINA PECTORIS, in its typical form, is in my experience a very rare disease. Pseudo-angina, or what resembles it at times, cardiac asthma, is not infrequent. Although angina pectoris is described among the neuroses of the heart by authors, this view in my judgment is frequently erroneous. In the great majority of instances where angina is present there exist also organic changes of the coronary circulation, of the cardiac muscular fibres, or a lesion of the aortic orifices.

I should be loath to admit, except in a very exceptional way, the existence of true angina purely of neurotic origin. On the other hand, pseudo-angina is very frequently of this provenance, accompanied by symptoms of flatulent dyspepsia. It must be understood, however, that there are cases on the border-line in which, during life, we have great difficulty in pronouncing as to what symptoms are of nervous origin and what are clearly due to organic changes of the heart or arteries.

In many such instances, unfortunately, even if death occurs, we are not always able to obtain verification of clinical facts by the results of an autopsy. Hence, certain theories are brought to the front which have no basis in actual, reliable observations from the dead-house.

One statement is certainly true of angina, viz.: that it has usually connected with it an element of spasm or sudden intracardiac pressure which separates it notably from conditions which, in many ways, are similar, and yet from this stand-point differ manifestly in a greater or less degree. Of the truth of this statement we shall be convinced later.

In general, it may be stated that pain, properly speaking, does not characterize organic disease of the heart as we commonly meet it. Of course, there may be more or less præcordial anxiety or oppression, or there may be pain in the vicinity which is of stomachal or hepatic origin; but acute cardiac pain is very rare. For this reason it has become almost an axiom for clinicians to say when acute cardiac pain is complained of, it is more than likely no organic disease of the heart is present. Perhaps this affirmation is too positive or dogmatic, since I am confident functional distress may occur which is dependent upon obvious tissue changes.

In true angina the pain is very characteristic, and as I have said, ordinarily means organic changes of the heart structure. First of all, the pain of angina is marked by its great intensity. In no other disease, perhaps, is this so true. When the anginoid attack occurs, if the patient is walking or exercising in any way, he stops immediately and holds himself as quiet as possible, only taking hold almost involuntarily at times, so great is his distress, of the nearest object which will give him support. Otherwise, he may merely stand rigidly, having come to a short stop, with his arms lying unconsciously by his side, or else one hand is pressed firmly in the præcordial region, as if to ameliorate in a measure the subjective agony by so doing. During the attack the pectoral and other muscles of respiration scarcely move, and, indeed, breathing comes almost to a stand-still for a while. In this we perceive at once the great dissimilarity with an attack of cardiac asthma, in which the efforts to breathe are so forcible and striking. And yet there are numerous instances in which the cardiac asthmatic features are most notable and where the "*angina sine dolore*" of Gairdner is also present,[1] as Osler states, after a masterly summary of the differential diagnosis of these two states, "when we recall to mind the features of the attack in cardiac asthma and in certain anginal seizures, the similarity of the condition, as Huchard remarks, to an acute emphysema, the views of Von Basch[2] appear to possess at least a reasonable probability" (p. 85).

The locality of the pain in angina is not always the same. Frequently it is located over the præcordial region. At times, however, it may be situated in the upper portion of the chest, or again, but in relatively few instances, it may stretch like a heavy bar across the xiphoid cartilage and the adjacent structures. Under these circumstances the pain may radiate directly through the chest and be felt even in the back. The pain has been likened to a heavy weight or crushing pressure, as though the thoracic parietes must almost meet. The pain is also said to resemble that of some terrible griping, as though the heart were firmly held in the clutch of some terrible monster. All these sensations, and numerous others, have been described and dwelt upon by writers, who doubtless have used their imagination at times to supply the descriptive powers of the patient. Suffice it to add that the pain is of a frightful sort and quite unlike any other we are familiar with. Often the radiation of the pain is toward the left arm, and in that case is usually carried through the forearm also and to the fingers. The ring and little fingers are said to be usually affected. Very rarely the pain extends to the right arm. When it does it radiates likewise in one or other direction mentioned.

According to Broadbent, the pain of angina often originates in the left wrist, and from there travels upward through the left arm and toward the chest—or again, as Osler states, although originating in the chest,[3] "was felt very severely about both wrists." This must be an extremely rare expression of the pain. I have never met it, nor do I find it mentioned by others. It is clear that the relations of brachial symptoms[4] (neuralgia) to angina pectoris are various. Sometimes, though infrequently, there are no pains in the arms, even in quite severe attacks of angina. Again, the brachial symptoms are very prominent, begin the attack of angina, and last longer than it does. The fact is, however, that the description of pain and its radiation, especially where it has the remarkable intensity of that belonging peculiarly to angina, must be somewhat inaccurate at times. In any event, it could only be obtained after the attack has passed, and I am inclined to believe that only exceptionally the patient could give any graphic and truthful description of it. What is literally true is that the patient has the impression vividly marked of impending dissolution, and it is this sense, together with the character, site, and evident intensity of the pain, which are almost pathognomonic of angina.

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[1] Osler. Angina Pectoris and Allied States, p. 82.

[2] These are: "Cardiac dyspnœa follows swelling and diminished elasticity in the lungs."

[3] "Angina Pectoris and Allied States," p. 42.

[4] Boston Med. and Surg. Journ., March 14, 1901, p. 256-57.

I know of few things more remarkable in descriptive medicine than the account given in the life of Dwight L. Moody by his son, of the anginoid attack near the close of the life of the great evangelist. In this case there was no terror or mental distress, as I believe, because his faith and works fixed him, as it were, on a rock. But in very many instances there is unquestionably great terror and mental distress. This is pictured often in the countenance which has that gray, ghastly, drawn look which once seen leaves an indelible impression upon the observers who may be near. The different radiations of the pain in angina are, no doubt, reasonably explained by the position of the cardiac plexus. The site of this plexus near the heart would serve to strengthen this view. In addition, we have its divisions and communications which appear to justify this interpretation.

According to one eminent observer, the pain originates probably in the central nervous system. This writer also explains its radiation and extension by affirming that it proceeds from the spinal cord. One thing is pretty certain, if we may judge by the few thorough observations we have recorded of these cases, and it is that there is no pressure outside of the heart on the plexus from any form of aneurism or other kind of tumor. While the heart may or may not be enlarged according to circumstances, this enlargement, even though present, does not explain rationally the anginoid symptoms.

Cardiac hypertrophy and cardiac dilatation are very frequently met with, and yet in the vast majority of these cases there are no anginoid symptoms properly speaking. Leaving aside these instances, there are a few where the heart is seemingly of normal size and volume, and we must, therefore, seek an explanation of anginoid pains in some other direction. Even in acute cardiac dilatation, no intense pain is felt, and yet we should have in just such cases pressure on or distention of nervous fibres under the endocardium. Moreover, this pressure or distention must be very much greater in patients thus affected than in those suffering with angina where no similar condition exists. During the attack the patient is usually very pale, and the pallor has a certain gray, ashy hue which is indicative of the serious condition which occasions it. Together with this pallor there is extreme weakness, and a faint feeling, which cannot be resisted, overwhelms the individual who is attacked. The pulse shows by its character, oftentimes upon what this weakness in part depends. It is frequently small, feeble, irregular, as though the poorly acting heart could not send the blood to the extremities. Again, singular to say, it is almost unchanged, at least so far as we can appreciate by our tactile sensations. In the former instance, it is probable that there is present a spasmodic contraction of the peripheral arteries; in the latter, we must assume that no such spasm exists, or, indeed, that arterial changes are so advanced that no marked impression is made upon their contractibility even by the most intense pain and disturbance of the central organ of circulation.

Frequently an attack of angina terminates by a sudden explosion of gas from the stomach. Hence, it is often stated and familiarly accepted that flatulent dyspepsia is an immediate and efficient cause of a true attack of angina. In my experience this is scarcely true, and I am more inclined to the belief that it is especially in cases of *pseudo*-angina that we should expect to find symptoms of stomachal weakness or intolerance. While admitting this, we should also not completely ignore the fact that the stomachal conditions which occasion flatulence may at times appear to be of considerable importance, taken with other exciting factors, in bringing on an attack. There may be, as Broadbent points out, a certain sympathetic relationship between the terminal fibres of the vagi in the stomach and those in the heart. Many facts would serve to demonstrate this possibility. Certainly, even in cases of marked cardiac weakness, where there has never been a true anginoid attack, dyspepsia of an acute and very distressing type will frequently follow undue fatigue of any sort or any severe shock to the nervous system such as distressing or alarming news may readily excite.

A phenomenon which is somewhat curious is the fact of an intense desire to urinate during the period of an attack, even though the effort is vain, simply because the bladder is frequently entirely empty. This statement may not invariably be true. I have known many a time emotional excitement to prevent absolutely for a while the contractile power of the bladder being exerted, and where, as was proven later, the bladder contained a considerable quantity of urine. It requires a very slight degree of annoyance or mental disturbance in men past middle life to prevent frequently their power to void their urine. Of course, the contrary of this is true, especially among women of a neurotic type, and who are still relatively young. The quality of urine of low specific gravity passed by them at times, in a very brief period, is often very great. In the differential diagnosis of true angina with pseudo-angina, this point should be borne in mind. During the attack where it is severe, perspiration will flow almost constantly from the patient. His face and neck and hands may be covered with it. It is cold and clammy, and lends additional significance to the gravity of the other symptoms.

The time during which an attack lasts is very variable. Sometimes it is over after a few seconds, although during this short period the agony is fearful. Again, the attack is prolonged for several minutes. Some authors state that the attack may occasionally last throughout an entire night, and that during all this time the patient is unable to move at all on account of the intense pain, and, moreover, is perspiring profusely the entire period. I must confess that I have never seen any attack of this sort, and am inclined to consider them very infrequent.

It is highly probable, moreover, that in a case of true angina depending upon advanced degeneration of the coronary circulation, which would probably be present under such circumstances, the intense pain of anginoid character would terminate life more rapidly. Here, again, I should be disposed to hold the view that a neurasthenic or hysterical element was present, which gave strength and exactness to the true diagnosis, viz.: pseudo-angina.

It must be admitted, of course, that there are instances in which the attacks are certainly anginoid in character, although they do not reach their complete development. This fact may be explained by stating that the patient, having suffered from attacks previously, so soon as he fully appreciates that one is coming on simply stops still and avoids all possible exertion until the attack has completely passed away. In these examples there may be pain in the chest, but without radiations toward the arms or fingers. Of course, if the pain is diminished in violence, there is less dread attached to the seizures, and the patient does not expect to die at any moment. Broadbent and others speak of anginoid attacks *sine dolore*. In such instances the chief danger arises from a syncopal attack in which a patient may suddenly expire. It may be that these attacks had been originally painful, and it was only subsequently that they lost this characteristic feature entirely. I should expect, in such an instance, to find at the autopsy either marked fatty degeneration of the heart walls independent of coronary changes, or else advanced aortic regurgitation. Wherever the coronary circulation is suddenly obstructed with an embolus or thrombus, the breast pang seems to be almost an invariable accompaniment. In those cases where the attack has evidently been brought on by exposure or exertion, it does not usually last long, and when the accidental occasion of the attack has disappeared, the seizure itself is apt to dissipate itself rapidly. Wherever the attack comes on spontaneously, as it were, without any accidental efficient cause being evident, it is apt to last a longer time, and only to pass away little by little and slowly. Occasionally these attacks are the most alarming in reality, and herald a fatal termination in the not distant future.

Among the causes which act efficiently in bringing on an attack of angina are primarily exertion. We are apt to say over-exertion when the attack has taken place. This over-exertion may be a brisk, rapid walk, or the patient may be walking leisurely and without effort when the seizure occurs. Usually, however, it is when a walk has been prolonged and there is already a feeling of fatigue that the angina is felt. It has been noticed that whenever exertion takes place soon after a meal an attack is more apt to occur. It may be because digestion, if slow and torpid, is thus interfered with, and gases which are generated and accumulate in the stomach press against the diaphragm and indirectly against the heart, and thus, by causing some displacement of this organ, may occasion notable interference with the circulation.

One reason, no doubt, why attacks occur at times during sleep, is because flatus is prone to accumulate in the stomach and intestines during sleep, and considering this together with the fact that in the horizontal position we have more pressure of the abdominal viscera upward, we realize readily conditions which are powerful in causing distress. The liability, under these circumstances, to an attack is increased notably by a feeble circulation. Moreover, as we know, the circulation is always less active in repose, and this state is what prevails during sleep. A loaded rectum is, also, a condition to be avoided, and anyone subject to constipation must see to it that the bowels are properly evacuated. The distended bowel may perhaps act in a reflex manner, as well as by direct pressure.

The influence of cold is sometimes very evident. This is particularly true when a patient is walking against a cold wind. Nothing more is required than this sometimes to precipitate an attack. On the other hand, mild, warm weather is conducive to well-being, and sufferers from angina will often escape attacks during long periods when the weather is free from rapid changes and remains relatively balmy. It is essential at night for a patient to see that the bed is comfortably warm and that no chilling of the surface ensues; otherwise, an attack will often follow. This precaution may be readily attended to with a hot-water bottle or heated bricks. Gentle friction of the surface of the body, perhaps, before the patient retires, is also a proper precaution to take. The wearing of long woollen stockings, and particularly those which are somewhat loosely knit and allow free transpiration, is especially desirable, so as to keep the extremities suitably warm. I know of no small detail so important as this in all affections in which the circulation is notably impaired, and, of course, it becomes doubly imperative in warding off painful attacks which are too frequently occasioned by local chilling of the feet.

In view of the fact that dyspepsia is such a frequent symptom of angina, and appears as an efficient cause, in the judgment of a few writers, quite as often as an effect, it is important to avoid all late or too abundant dinners. The food at this meal should be of the simplest kind, and no overloading of the stomach should be permitted. In a similar way, no sauces, condiments, or insufficiently cooked food should be tolerated.

Whenever an attack has occurred, it behooves the patient to be more than usually circumspect in all his doings, not to bring on another one. This is especially true of any exertion which seemingly has been the direct cause of an outbreak. And yet this counsel is sometimes almost unnecessary, because the patient's own feebleness, which follows a primary attack, will compel him almost to walk very slowly and deliberately, even if he walks at all, for some hours or days subsequently. No doubt these anginoid attacks would not occur if the heart had sufficient reserve force to respond adequately to the call made upon it. Unfortunately, it has not, and it is therefore evident, in many instances, that the attack is directly occasioned when we reach final causes, by the manifest inability of the heart to respond to the call made upon it for increased vigor.

Angina pectoris is not necessarily connected with any special lesion. Practically, it is almost unknown to have either stenosis or incompetence of the mitral valve appear as a direct, efficient cause of it. It is true, however, when there have been several attacks of angina, it is not infrequent to observe mitral incompetence arise subsequently. In some of these instances it has been noted, where aortic incompetence already existed, that this affection was ameliorated as regards its symptoms, and that the anginoid attacks also became less severe. The explanation appears to be in the lessened blood pressure thus brought about, as shown in the arterial pulse and in the diminished accentuation of the aortic second sound. Musser[1] has insisted upon the importance of this finding, and has reported several examples in his own experience. Broadbent[2] has also specially emphasized similar instances.

Angina is frequently connected with fibrous myocarditis, and at the autopsy such organic change in the heart muscle is apt to be found. So usual is this condition that Gibson states it is almost a surprise not to find it. When fibrous myocarditis is noted, it is frequently accompanied with evidences of arterial degeneration. The coronary arteries are usually implicated. Especially is this true where aortitis is present. The lesion may be limited to their origin, which is sometimes narrowed and thickened. The arteries may also be affected in a considerable extent, and the organic changes may be considerably advanced. Occasionally they have merely lost elasticity; in more pronounced alteration they may have become markedly atheromatous, or, indeed, calcified.

According to Douglas Powell, fatty degeneration of the heart walls often exists. Sometimes, indeed, the heart is so much degenerated that it is easily torn, and the finger sinks into it on slight pressure. Sometimes, to the naked eye, the fat exists only in patches, affecting merely the papillary muscles or different areas of the ventricles. Even in these instances, however, if we make use of the microscope, we are apt to find considerable degeneration of the walls, where there has been no real change of coloration. In a few instances the microscope shows almost complete disappearance of the muscular fibres.

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[1] Transactions of the Association of American Physicians, vol. x. p. 85.

[2] British Medical Journal, 1891, vol. i. p. 747. Quoted by Osler.

As a concurrent condition with fatty degeneration, we discover more or less advanced changes of the coronary arteries, very similar to those already mentioned in connection with fibrous myocarditis. These evidences of fatty degeneration are particularly found, of course, where the fatal termination appears to be intimately dependent upon the previous anginoid attack. It should be remarked in this place that we often have both anterior conditions, viz.: that of fibrous myocarditis or of fatty degeneration, without having attacks of true angina. According to Gibson, the relations of angina with endocardial lesions is not so distinct. It is true, of course, that degeneration of cardiac walls may often cause it, and, therefore, it is frequently found at the autopsy. The earlier writers, like Morgagni, certainly attached angina directly to the existence of aortic disease, and in one of Heberden's cases, where the autopsy was made by John Hunter, this affection is duly recorded. On the other hand, we know that the most advanced changes with ossification may exist at the aortic orifice, and yet there may be present during life no morbid symptoms at all.[1] These instances must be somewhat exceptional. What is true is, where aortic lesions have been proven at autopsy, often pain has been noted prior to death. No doubt, this pain has been in part due, at least, to interference with the coronary circulation, caused by accompanying aortitis, with which there may be also a certain degree of dilatation, or, indeed, a sacculated aneurism. Arterial degeneration, especially arterio-sclerosis, is often adjoined to attacks of angina. Where the coronary arteries are degenerated, and where the angina is seemingly dependent upon this condition, we should not lose sight of the fact that the other arteries, being degenerated, are also doubtless contributory. The affection of the coronary arteries through sclerosis and consequent narrowness, prevents a sufficient blood supply reaching the heart, and hence interferes with its nutrition. A thrombus or embolus may obstruct the vessels, but it is doubtful whether one or the other of these conditions causes angina (Gibson). One thing is sure, viz.: we often find calcification of these arteries without previous anginal attacks. Adherent pericardium may be found, but does not occasion anginal attacks unless accompanied by a lesion at the aortic orifice.

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[1] Semple, p. 104 et seq.

Angina has been observed following injuries to the chest walls. In these cases the aorta may have been affected. Broadbent states its presence occasionally in malarial fever. It may also be present in advanced diabetes where the arteries may become thickened, thus giving rise to increased tension, and followed by attacks of true angina. It is not infrequent to find anginoid attacks occasioned evidently by the presence of the gouty poison. In these cases the prognosis is only serious where the intracardiac changes are already advanced, as shown by the weakness and irregularity of the heart's pulsations. In some of these cases we find notably fibroid myocardial changes in patches or disseminated. Even in these instances, prior to death, there may have been few or no threatening cardiac symptoms. In a few rare cases neuritis of the cardiac plexus and also of the phrenic nerve has been noted (Lancereaux, Peter) where anginoid attacks have occurred. The view of Semple is, indeed, that angina considered as an idiopathic disease is connected with an affection of the pneumogastric or phrenic nerve. Of course, it is difficult always to pronounce what the precise structural changes are. Still, they are doubtless present, and later will be discovered. Meanwhile we are forced to rank a few such instances among the "neuroses." Frequently, doubtless angina is associated with minute structural changes which only subsequent close investigation will determine. Flint is evidently of the opinion that the connection between angina and organic lesions of the heart is rare. Thus he has only observed fifteen cases in 388 cases of the latter. Again, Flint says that in ten years he has noted only four cases of true angina, that is to say, where the disease was unconnected with cardiac or aortic lesions. It is clear that in all instances where there is present an organic lesion of the heart or arteries, whatever suffering, if any, the patient may experience should be directly explained by them. This leads to the conviction that only those cases in which no such lesion is discoverable should be ranked among the true cases of angina. As a rule, when sudden deaths occur in what has been called angina, pathological lesions sufficiently explanatory are found at the autopsy.

The condition of the heart during an attack of angina has been believed to be one of spasm. At least, this is the opinion of some writers. It is certainly true of Heberden, who first so accurately described these attacks in his commentaries. This does not seem to be altogether a tenable opinion, if one has regard to the fact that the heart has rarely been found thus contracted.

Usually the heart stops in diastole, and is found after death in a relaxed condition. Again, during life, while the pulse at the wrist is sometimes irregular and weak, it never disappears entirely, which it certainly would if the heart were in a state of forcible, spasmodic contraction.

It has been supposed that the heart during an attack presented a sort of hour-glass contraction not dissimilar to that of the uterus. This may be, and yet it would be difficult to prove. Broadbent confesses in this connection that he has a very imperfect notion of what the condition of the heart really is during an attack. The evident fact is that during a paroxysm of angina stress is put upon the heart to which it is quite unequal to respond, and thus it shows its considerable lack of power. Often the stress put upon the heart is due to the continuous high tension of the peripheral arteries. Occasionally, however, there is low tension in the peripheral circulation, and in these instances, if there occur a sudden general arterial spasm, the amount of work thrown upon the heart becomes rapidly much greater, and consequently the heart shows relatively greater distress than where the peripheral resistance is continuously high and exaggerated. In those cases where there is marked and continuous high tension in the peripheral arteries we might suppose that pain in angina was explained by greater pressure thrown upon the heart. This can scarcely be true when we consider how many such cases escape any such pain. Again, in acute dilatation of the ventricles, we have great pressure brought upon the heart walls, and yet no pain results. Neuralgic predisposition is occasionally given as an explanatory cause. This is scarcely true if we mean by that an acquired or evident neurotic tendency, since this disease occurs more frequently with men than with women. It would seem as though from the fact that when the attack occurs immediately, the patient stops doing anything he is occupied with, or exercising, if that be what he is about, that there is a certain pre-ordered protective arrangement internally to guard against these outward manifestations of man's life. (Broadbent.)

Pain, according to Bramwell,[1] is due to irritation of the nerve terminations in the walls of the heart itself. He admits, however, that the theory of irritation due to spasmodic contraction is plausible, and compares this opinion with what occurs in the calf muscles, spasmodically contracted, in ordinary cramp.

Attacks of false angina often resemble those of the real kind. Sometimes the description of the attack by friends will enable us to reach a correct diagnosis. If the patient becomes pale, anxious, and shows signs of great distress, it may not of necessity be true angina. If, on the contrary, there be no such changes evident, we can be very sure that it is only an attack of pseudo-angina. There is much unreliability in the patient's accounts, mainly because they are apt to read up about these attacks, and often give an exaggerated idea of their own sensations. Age will throw some light upon the diagnosis. Under forty years of age in the male, angina occurs very rarely unless there be a pronounced lesion at the aortic orifice or aortitis. In females it is rare at any age, although attacks of pseudo-angina are not infrequent with them, especially those of an undoubted neurotic temperament. Heberden, for example, in speaking of the cases observed by him, being in number over 100, states that three occurred in women, one in a boy, twelve years of age, and the others in men near or over fifty years.

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[1] Diseases of the Heart, p. 676.

Usually the first attacks of angina occur during physical exertion. Later on they may come on severely and more readily, and then we may make the diagnosis surely, even though the determining cause is slight. The physical examination will reveal, in case of true angina, the changes mentioned in the aorta or at aortic orifice. Where the attack at first comes on without exertion and at a fixed period after ingestion of food, it would seem to be of digestive provenance or pseudo-angina, and ordinarily due to a dyspeptic attack. Unless history, nature, and onset of attack all concur together with physical signs to establish diagnosis of true angina, we should lean strongly to diagnosis of pseudo-angina, and almost invariably, if we can discover any facts to support this diagnosis. If we leave out attacks of pseudo-angina which are evidently neurotic or of hysterical nature, we can usually find in some digestive disorder, particularly of the stomach, a sufficient explanation of them. There are eructation, marked flatulence, pain, occasional attacks of nausea or vomiting, which all point in this direction. A combination sometimes found is that of dilatation of the stomach, with high arterial tension. If the heart be affected in these cases and they are improperly treated, a fatal result may not infrequently follow. If, for example, digitalis or nitroglycerin be alone used, or the Schott treatment advised and carried out without any care of the stomach, and especially if the subject be old, such a denouement may not be a surprise.

The prognosis of true angina is often uncertain; and yet we have certain conditions which guide us to make it correct. We should estimate carefully the relative predominance of the two factors often producing it—on the one hand, degenerated heart walls, on the other, vascular changes. If there be high arterial tension present, and if at the same time the heart action is forcible and the aortic second sound marked, we may hope by proper treatment to modify these conditions for a while with the use especially of nitroglycerin and the nitrites. Again, if over-exertion and excitement bring on the attacks, or if flatulent dyspepsia be a decided and powerful influence in producing them, we should hope to avoid with care and treatment their natural outcome.

On no account should the patient walk hurriedly, especially in going up hill. He should also never take even a moderate walk until a certain time had elapsed after his last repast.

Attacks of angina which accompany aortic disease may last a considerable time without bringing on a fatal result if carefully watched and guarded. The worst cases are those which recur in the night or at times where no accidental cause is present and avoidable which occasions them. Again, if examination is relatively negative, if the heart is of normal size, without manifest lesion of any kind, and yet its action is feeble, its impulse scarcely felt, and the pulse usually, if not invariably, of low tension, these give great anxiety by reason of the vagueness and uncertainty as to the conditions which may be present and at any time become imminently threatening.

The apparent severity of two attacks may be similar, and yet the relative danger of them may be absolutely different. It is difficult, therefore, at times to make anything like a sure forecast. Of course, where there are pronounced cardiac and arterial changes, and where, in addition, heart failure has followed hypertrophy, the outlook is assuredly very grave indeed. If, at the same time, aortic regurgitation also be present the prognosis becomes even more serious. In a similar way, if chronic renal changes exist the future of the patient must appear dark and imminent. Gibson states that the prognosis of those affected with *fatty* degeneration is *far less* serious than the preceding. Provided always the external and avoidable causes of aggravation are prevented, such patients may often live many years. Of course, toxic angina is far less grave. As a rule, with the removal of the cause the case becomes curable. In neurotic cases, while we should expect frequent recurrences of the paroxysms, it is wholly improbable to have a fatal termination.

In this category may be placed frequently the so-called idiopathic cases. They are often extremely painful, but as no incurable lesions exist, they tend to improvement or recovery if properly managed. Of course, we should be careful in making even in these cases too favorable a prognosis, since there may be some underlying structural change of the heart walls or coronary arteries which, during life, could not be determined. There are unquestionably, according to Semple, certain cases of pure angina in which the autopsy reveals no organic changes.

The treatment of angina depends upon what is the apparent or obvious cause. In many instances, owing to the difficulty of tracing accurately to what the attack is primarily due, our treatment must be essentially empirical. First of all, we must consider the general health, and from this point of view our treatment should be hygienic. The means at our command are here what pertain to air and light, rest and exercise, food and drink. After these have all been inquired into and regulated, as far as may be, we naturally seek for the proper medicinal remedies to meet the indications of each special case. In general, also, the efficacy of our treatment will depend much to what degree we may be able to relieve peripheral resistance to a heart frequently weakened. If, perchance, we find between the regular pulse beats, evidence of increased tension to our tactile sensations, we may often reduce this by appropriate remedies. Still, in order to recognize it, we must at times examine the heart and arteries at different periods, before and after exertion. Not infrequently the arteries are notably degenerated, hard, thickened, tortuous, and even calcareous. We can then do little to affect them directly. Yet, the capillary system, in which there may be notable resistance without excessive changes, and which has caused in a measure the arterial and cardiac changes, may be still favorably influenced by appropriate drugs, and account should be kept of this fact. In gouty conditions the peripheral circulation may show increased tension, although not visibly degenerated, and this condition, of course, may be favorably influenced by appropriate medication.

In these latter cases the ordinary treatment with a mercurial, followed by a saline, once or twice a week, will lower arterial pressure. Between times the use for a while continuously of iodide of potash and colchicum may be of signal benefit. The employment of bitter tonics, if the indication presents, and the proper regulation of the diet is of course useful According to Powell, hop, columba, and chiretta are better tonics in these cases than quinine and strychnine. In the "neuralgic bouts," to which they are prone, he praises quinine and phenacetin. Where angina occurs with marked aortic disease it is difficult sometimes to know to what extent we may be able to help the attacks by reducing tension of the pulse. Where the pulse remains feeble between the attacks, and the heart has a weak impulse, we should carefully endeavor to help with cardiac tonics, but frequently we can be of little real service in view of the pronounced degenerative changes present in the heart and arteries. Occasionally arsenic, combined with iodide of potassium and nux vomica, is useful where the arterial tension is not too pronounced. Preference may be given in many instances to the sodium salt of the iodide, both between and during the attacks. According to Schott, it is less prone to cause heart failure; but even this salt is "apt to destroy the molecules of albumin" if continued too long or in increasing doses. Milk is the best menstruum for either salt, as in this way stomachal intolerance is less likely to occur. In these and other cases we should try to preserve the use of the nitrites and nitroglycerin for the attacks. Formerly the diffusible stimulants, like brandy, ammonia, lavender, camphor, etc., were much used for these attacks. Now they are almost abandoned for nitroglycerin and nitrite of amyl. These latter are particularly useful in relieving pain, and to accomplish it they dilate peripheral arteries. Nitrite of amyl by the rapidity of its action is preferably employed. Nitroglycerin and the sweet spirits of nitre produce similar effects in different degrees.

All of these are free from dangerous effects, as a rule; not so of nitrite of sodium, which may produce alarming results (Gibson). Nitrite of amyl and nitroglycerin dilate arteries, increase frequency of pulse and respiration, and reduce irritability of the nervous system. Where increased acceleration of the pulse and respiration are already present the nitrites must be employed with great care, as they might possibly cause greater distress. While they are said to be heart stimulants, they mainly cause relaxation of the arteries and also of the cardiac muscular fibres (Broadbent).[1]

The nitrites have their drawbacks also in the fact that patients find so much relief from their use that they use them too frequently and injudiciously. A word of warning should be thrown out because life is sometimes shortened by their inconsiderate use. Glycosuria has been produced by them, it is stated. In many instances the nitrites are less useful than iodide of potash. Nitrite of amyl may be carried about with one so as to be used immediately. The nitrite of amyl in glass globules, of 3 to 5 minims, may be in a silk bag and broken upon a handkerchief and inhaled as required. The nitroglycerin tablets, one one-hundreth of a grain, may also be taken in doses of one or two when attack occurs. They do not act as rapidly as the nitrite of amyl, but their effect is more prolonged, and on that account may be more valuable in certain cases.

Some cases, however, are not relieved by nitroglycerin tablets and are relieved by nitrite of amyl. According to Broadbent, such cases have seemed to him to originate in the right ventricle.

It is the belief of Dr. B. Addy[2] that we have in erythrol tetranitrate a remedy superior even to "nitroglycerin," its effects being very rapid and more lasting. Tablets of one-half grain each were given by him twice or three times a day. They did not cause headache, and the remedy soon checked the attacks. It is true, the patient died after a fortnight of syncope, but during this period great relief from suffering was experienced.

Sometimes, where the heart is weak and the nitrites do not relieve, although they may relax the peripheral circulation, we must recur to the old stimulants. In addition, a turpentine stupe, or mustard leaf, or poultice may be applied over the chest and will occasionally afford a measure of comfort. Whenever these local applications fail, great relief is obtained from a hot-water bag at a temperature of 140° F. to 170° F., "moved with light touches over the whole chest."[3] If, despite all this, the attack is prolonged and unrelieved, we must give a hypodermic injection of morphine and atropine, using at first small doses, and later, if need be, becoming bolder, and using larger doses. It is well to make injections deep in the muscle, where the circulation is more active than in the cellular tissue under the skin. In some instances, we should recur to chloroform inhalations as being the only hope of relief to the patient. At times they are undoubtedly dangerous, and especially is this believed to be true if fatty heart is present. As a matter of fact, however, fatty heart cannot always be diagnosed with accuracy. The apex may be strong and the pulse regular and good, and yet fatty heart may exist, and sudden death follow. Again, moreover, it has been shown that chloroform may be given safely where fatty heart later is known to exist by the revelations of the autopsy. We must relieve intense pain, however, even if there be risk, and it can only be done at times by such agents (Balfour).

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[1] This opinion about heart fibres I do not share save very exceptionally.

[2] British Medical Journal, May 6, 1899, p. 1089.

[3] Lancet, September 8, 1900, p. 726.

In cases where there is marked heart failure, ether or brandy should be employed hypodermically in doses of ʒss to ʒj. To each hypodermic injection one or two tablets of one one-hundredth of a grain of nitroglycerin may be added. The latter should be employed with caution, however, as occasionally considerable soreness and even ulceration of the skin may result.

Theodore Schott does not value very highly digitalis or strophanthus in cases where the heart requires stimulation, even in uncomplicated forms of angina pectoris caused by sclerosis of the coronary vessels.

Oxygen inhalations are often also useful, not only to satisfy the air hunger, due to obstruction of circulation in the lungs, but also to stimulate cardiac circulation and help nutrition of its muscle, and thus get rid of effete material which interferes with proper metabolism (Powell). In these cases the oxygen must be used with a funnel near the nose and mouth, so that it may be inhaled frequently and without effort. As corroborative of the extreme value of inhalations of oxygen in the treatment of some severe cases of angina pectoris, I would refer to one recently reported in the *British Medical Journal* for December 1, 1900, p. 1568.

Rest in bed for a time is often desirable after acute paroxysms have passed, but later it is useful, as far as possible, to get the patient back to his ordinary life with judicious restrictions. The same rules apply here, however, as in other heart affections. We must remember, also, that exertion which one day may seem all right, another day may cause distress and oppression. This is one of the objections to Oertel's system of treatment (Broadbent).

Physical therapy is undoubtedly useful in some instances, but it must be utilized with great care. This counsel pertains particularly at the present time to the treatment as instituted at Bad Nauheim, where the resistant movements in conjunction with carbonic baths are practised. In advanced arterio-sclerosis every increase of the blood pressure which is the result of this treatment might lead to fatal consequences (embolism, apoplexy, rupture of aneurism of heart, or aorta). " Advanced sclerosis is, therefore, a contraindication for this treatment" (Schott). The value of many medicaments in angina comes from producing low blood pressure. The balneological and gymnastic treatment exercises a tonic influence, and " by strengthening heart muscle, as well as by acting on cardiac nerves, distressing symptoms of angina are either removed or reduced" (Schott).

Guidance should be had in regard to the bad effects of winds, great heat or cold, or rapid changes. Also, an atmosphere heavily laden with moisture is injurious. Internal conditions of dyspepsia and constipation must be warded against. A great deal of tact and good judgment are required, and the patient's disposition should be thoroughly known. Rest, particularly after meals, should be insisted upon, as patients are particularly liable to attacks at these times.

In general, supervision and counsel must be employed about exercise. Where an attack has lately occurred, it is wisdom to refrain from exertion for a while, especially if the heart is weak and fluttering, and afford it time to re-establish itself.

THE PATHOLOGY OF THE HEALED FIBROUS ADHESIONS OF THE PERICARDIUM.

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In a total of 1048 autopsies upon the bodies of adults, the records of which are among those of the Pathological Laboratory of Rush Medical College (in affiliation with the University of Chicago) and of the Cook County Hospital, some inflammatory change was found in the pericardium in 128. These changes included everything in the nature of a pericarditis, from the frank serofibrinous and tuberculous processes to those with but a thin film of fibrin over a small area, or a fibrous band uniting the parietal and visceral layers at some point. Of these 128 cases 57 showed changes that were essentially chronic, the lesion consisting of fibrous tissue uniting the opposed surfaces for a greater or less extent. These figures agree closely with those of Leudet,[1] who found in 1003 autopsies adherent fibrous pericarditis 61 times. The remaining 71 were acute, and represented all the conditions usually seen in acute pericarditis.

Of the 57 cases with fibrous adhesions the cause could be attributed to rheumatism in 8; in 6 the lesion was frankly tuberculous, as shown